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# Complete Remission in Three Patients With Acute Myeloblastic Leukemia by Administration of G-CSF Without Antileukemic Agents

**Masuhiro Takahashi,[1,*] Tadashi Koike,[1] Yoshifusa Aizawa,[1] Makoto Kashimura,[2] Kunihiro Hayatsu,[3] Ko-ichi Nagai,[3] Atsushi Abe,[3] Masaru Urushiyama,[4] and Kumiko Yagisawa[4]**

[1]First Department of Internal Medicine, School of Medicine, Niigata University, Niigata, Japan
[2]Division of Hematology, Matsudo City Hospital, Matsudo, Chiba, Japan
[3]Division of Internal Medicine, Niigata Prefectural Central Hospital, Joetsu, Niigata, Japan
[4]Division of Hematology, Sado General Hospital, Sado, Niigata, Japan

**We describe 3 patients with acute myeloblastic leukemia (AML), who received rhG-CSF for infections such as pneumonia or for prophylaxis of infection, and who achieved complete remission. They had not received any antileukemic therapy before or during the administration of rhG-CSF. These findings suggest the possibility that complete remission can be brought about by G-CSF itself in some patients with AML. Am. J. Hematol. 56:42–44, 1997.** © 1997 Wiley-Liss, Inc.

**Key words: AML; G-CSF; complete remission**

## INTRODUCTION

In this report, we present 3 patients with acute myeloblastic leukemia (AML), who achieved complete remission by the administration of G-CSF without antileukemic agents.

## CASE REPORTS

### Case 1

A 64-year-old male was diagnosed as having AML with trilineage myelodysplasia on January 18, 1988. He was treated with only rhG-CSF and antibiotics for severe pneumonia. In addition to an increase of neutrophils and improvement of pneumonia, platelets and hemoglobin began to increase and were almost in the normal range about 1 month after initiation of rhG-CSF. Bone-marrow examinations showed the disappearance of blasts. Bone-marrow smears demonstrated normal differentials without myelodysplastic features. Although rhG-CSF therapy was discontinued thereafter, hematological data were stable. Complete remission continued for 4 months, but leukemia relapsed in September 1989. The patient died in May 1990.



### Case 2

A 54-year-old male was diagnosed as having pneumonia on May 30, 1992. A bone-marrow examination demonstrated the proliferation of myeloblasts (61.1%) and a chromosomal abnormality of 47,XX,+8. RhG-CSF and antibiotics were administered. Antileukemic chemotherapy was not performed. White blood cell counts and platelet counts increased promptly and the pneumonia improved. A bone-marrow examination undertaken on June 19, 1992 revealed a decrease of blasts to 3.8%, but with the presence of the chromosomal abnormality. Although rhG-CSF was continued, a bone-marrow examination done on August 26, 1992 showed an increase of blasts to 68.4%. Antileukemic chemotherapy was started on September 2, 1992, which induced the relapsed leukemia to complete remission, showing a normal karyotype.

*Correspondence to: Masuhiro Takahashi, M.D., Department of Medical Technology, School of Biomedical Technology, Niigata University, Asahimachi 2-746, Niigata 951, Japan. E-mail: matak@clg.niigata-u.ac.jp

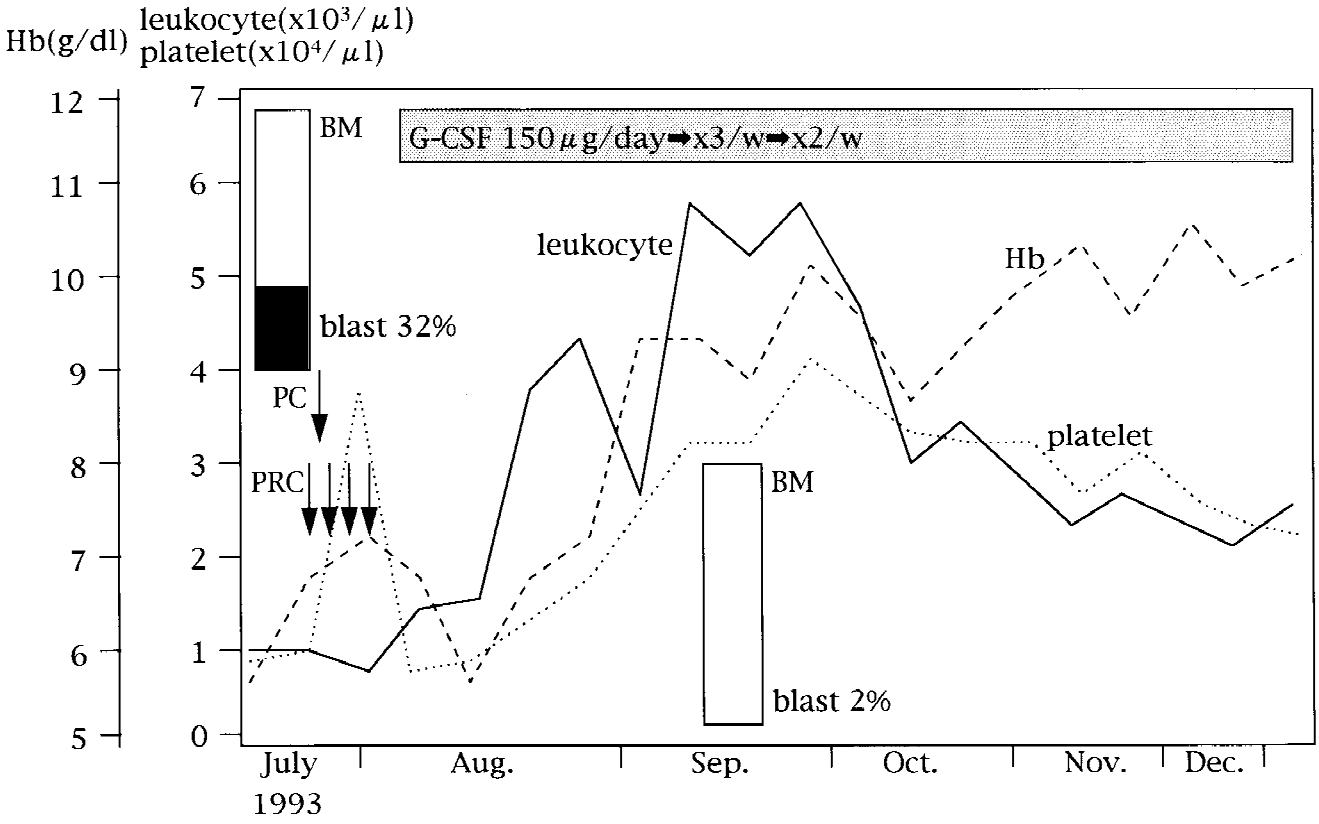


**Fig. 1. Clinical course of case 3. BM, bone marrow; PC, platelet concentrate; PRC; packed red blood cells.**

### Case 3

A 70-year-old male was diagnosed as having pancytopenia on September 24, 1991. Based on a bone-marrow examination showing an increase of blasts to 15.6%, he was diagnosed as having refractory anemia with excess of blasts (RAEB). Despite being treated with only vitamin D3, his RAEB progressed to acute AML. For preventing caries-related infection, subcutaneous administration of rhG-CSF was initiated on August 2, 1993. One month later, hematological improvement was observed and a bone-marrow examination demonstrated normal differentials but the presence of myelodysplastic features (Fig. 1). On March 24, 1995 he showed a proliferation of blasts (60%) in the bone marrow. The patient died of sepsis on September 29, 1995, despite antileukemic chemotherapy.

## DISCUSSION

There have been some reports that AML has gone into remission without antileukemic chemotherapy after the development of severe infection [1–3]. This phenomenon is thought to be associated with various cytokines generated by infection. Two patients with AML, who achieved remission by only the administration of G-CSF, have been reported [1,2]. Some patients with AML, who had received antileukemic chemotherapy but who did not achieve remission previously, showed findings of remission by treatment with G-CSF thereafter [3–6].

The present 3 patients with AML, who had not been treated with antileukemic chemotherapy, entered into complete remission after administration of G-CSF. Case 1, whose bone marrow showed myelodysplastic features at presentation, lost morphological abnormalities of marrow cells after gaining complete remission by G-CSF. On the other hand, in case 2, chromosomal analysis of marrow cells performed after achieving complete remission by G-CSF demonstrated the same abnormality as at presentation. In case 3 as well, remission marrow after G-CSF showed the remaining existence of myelodysplastic features. These findings suggest the induction of maturation of leukemic blasts by G-CSF. Case 3 is of interest for evaluating the role of G-CSF when going into complete remission. Demonstrable infection was not observed at the time of initiating G-CSF and for the duration of administration. The present 3 patients, especially case 3, suggest the possibility that the administration of G-CSF without antileukemic agents can prompt AML into complete remission in some patients with AML.

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